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The cerebral Windkessel and its relevance to hydrocephalus: the notch filter model of cerebral blood flow

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Background

Flow MRI studies have demonstrated that hydrocephalus is associated with significant abnormalities in intracranial pulsatility. A physiologically important aspect of pulsatility in the cranium is the Windkessel effect, the dissipation of arterial pulsatility rendering capillary blood flow nearly pulseless. We investigate the cerebral Windkessel mechanism using spectral analysis of pressure measurements in dogs.

Materials and methods

Carotid arterial pressure and intracranial pressure (ICP) in 12 dogs were measured during progressive withdrawal and infusion of CSF. Pressure waveforms were recorded and synchronized in time, and analyzed with Fast Fourier Transforms. Using an autoregressive moving average technique, the arterial/intracranial pressure transfer function was derived.

Results

The transfer function provides the frequency dependence of the efficiency of the transfer of arterial pulse waves into the intracranial pulse wave. The transfer function was characterized by a notch (i.e. a transfer for pulsatility from arterial to intracranial pressure) near the cardiac frequency of the animal. With increases in mean ICP, the depth of the notch decreased.

Conclusion

The transfer function measurements show that the cranium suppresses the carotid arterial pulse specifically at the cardiac frequency, allowing transfer of arterial pulsatility into smooth cerebral blood flow. The fact that the effect is diminished with changes in mean ICP implies that this suppression is impaired under altered intracranial conditions. We hypothesize that these results are the first preliminary evidence that the intracranial Windkessel mechanism may operate as a notch filter, a frequency-sensitive filter that suppresses a specific frequency of oscillation. The mechanical properties of the cranium are critical in providing the optimal operation of this suppression. Our spectral analysis suggests that the cerebral Windkessel suppresses the heart rate component of the arterial pulse, and may thus augment cerebral blood flow by transferring pulsatile flow into smooth flow. Elevation of ICP alters the mechanical properties of the brain and thus impairs this mechanism.

There is growing evidence that hydrocephalus is associated with significant abnormalities of intracranial pulsations, and pulsatile abnormalities may play an important

role in its pathogenesis. We present evidence that intracranial pulsatility is minimized via suppression of the arterial pulse wave in the brain. Hydrocephalus has been associated with spatial redistribution of pulsations (e.g., increased ventricular pulsations) and we have shown that changes in intracranial pressure may also lead to a "spectral" redistribution of pulsations (increased pulsatility at the expense of smooth cerebral blood flow). This observation may provide further insight into the pathogenesis of hydrocephalus.